

ORIGINAL ARTICLES

PSITTACOSIS

WITH REPORT OF TWO CASES

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DISCUSSION by Karl F. Meyer, San Francisco.

THE diagnosis of obscure fevers has been facilitated by a better understanding of the symptomatology and pathology of disease caused by certain incitants. The closer coöperation between clinician, clinical pathologic laboratory and, at times, the State Public Health Service, has made for efficiency. The common diagnosis of influenza with or without bronchopneumonia has served to screen more careful investigation of probable etiologic factors.

The following two cases of psittacosis (with recovery) are presented to demonstrate the ease with which the etiology of this obscure fever may readily be confused and mistaken.

REPORT OF CASES

CASE 1.—C. T. C. (65). American pet-shop dealer in Santa Barbara.

Family History.—Wife, age 62, shares the duties of the pet-shop. One daughter, married, is in good health.

Past History.—The patient has always enjoyed very good health, having had no serious illnesses nor operations. A mild bronchitic cough for many years, nocturia two or three times, and moderate overweight have been present.

Occupation.—The patient has been a dealer in birds for twenty-eight years, having been in Chicago prior to coming to Santa Barbara in 1912. He has been in contact with imported birds from South American ports as well as the Orient during many years of his life, and has related numerous instances of nursing sick birds, parrots, and parakeets coming in shipments to his shop. Never, however, has he had symptoms of illness comparable to those experienced at the present time.

Present Illness.—About June 14, 1935, generalized aching throughout the body, with malaise and anorexia were experienced. A few days later chills and fever were complained of. Nausea and vomiting followed with a persistent bad taste in the mouth. After June 17 there was a sense of fullness in the pit of the stomach, and no tolerance for food without vomiting was daily present. A dull aching pain in the upper abdomen started about June 20, not entirely relieved by emptying the stomach nor taking soda. A moderate constipation, slight hacking cough with mucopurulent expectoration were present. Restlessness, anxiety, and prostration were apparent.

Physical Examination.—June 22, 1935—revealed an elderly male, flushed, with blood-shot eyes, perspiring freely, and complaining of severe pain in the abdomen. The pulse rate was 80 per minute; respirations, 20; temperature, 103 degrees Fahrenheit; and blood pressure, 140/65. The eyes were red, and conjunctivae were injected. Except for marked tenderness in the epigastrium, with moderate abdominal distention, there were no abnormal signs elicited. Careful search for evidence of pulmonary pathology was lacking. All the symptoms and signs were referable to the abdomen.

Laboratory Tests.—June 23, 1935: Blood, 90 per cent hemoglobin; red blood cells, 4,620,000; white blood cells, 6,100; 82 per cent polymorphonuclears; 12 per cent small lymphocytes. June 27: white blood cells, 6,450; 85 per cent polymorphonuclears; 14 per cent small lymphocytes. Urine, specific gravity 1.028; trace

of albumen; trace of sugar; trace of diacetic acid. Widal, negative. Agglutination undulant fever, negative. Kahn, negative.

Course of disease and investigation: The acute illness warranted hospitalization, and the patient was admitted to the Saint Francis Hospital, Santa Barbara, June 23, 1935. Gastric lavage relieved the extreme nausea somewhat on admission. Colonic flushes, 10 per cent glucose in normal saline were administered supportively. On June 24 gastro-intestinal series was started. Preliminary fluoroscopy by Dr. Daniel Clark, roentgenologist, St. Francis Hospital, Santa Barbara, revealed an annular shadow in the mid-zone of the left lung. A portion of his report follows.

"There is a roughly circumscribed parenchymal infiltration adjacent to the left hilum and toward the anterior portion of the lung. This infiltration is about two inches in diameter and is fairly homogeneous in character. It shadows out toward the periphery. This infiltration is decidedly atypical. Its shape at first suggests a pulmonary metastasis, but its haziness of outline and the character of the density are more suggestive of an inflammatory process."

The hacking cough which was present at the onset increased in severity, and the sputum, at first mucopurulent, became purulent, at times flecked with blood. A specimen was forwarded on June 27 to Dr. K. F. Meyer, Hooper Foundation, San Francisco, approximately the thirteenth day of the disease. This material was extracted overnight in the refrigerator, centrifuged and the supernatant fluid injected into mice. On the ninth day one of the mice, obviously ill, was sacrificed. Typical lesions of psittacosis were present, and L. C. L. bodies—the causative elementary bodies of the virus—were demonstrated in the liver and spleen; smears thus establishing conclusively the diagnosis.

The patient was transferred to the infectious disease wing of the Santa Barbara County Hospital. The fever subsided by July 9, 1935, and he was released from isolation August 1, 1935, following the report from the Hooper Foundation that the sputum forwarded on July 20 failed to infect mice.

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CASE 2.—Mrs. C. T. C. (62). American, housewife, and wife of a pet-shop dealer with whom she shares the duties of the shop in care of pets.

Family History.—Her husband, C. T. C., had been ill since approximately June 14, and she had taken care of the store entirely from June 22 to June 27, when she took to bed, too ill to work.

Past History.—Not important, except that through the past twenty-eight years she also had cared for the birds and animals in the shop and nursed them in illness. She related many instances of sick parakeets and live birds she had taken care of in shipments from abroad when in Chicago.

Present Illness.—Approximately June 25, she developed chilliness, followed by fever. Weakness, aching through the body, lack of appetite, marked constipation, nausea, pain in the left chest and excruciating headaches early in the morning, marked the course of her illness. In view of the probable diagnosis of disease in her husband, she was admitted to the infectious disease wing of the Santa Barbara County Hospital on July 8, 1935.

Physical Examination.—Physical examination revealed a woman of sixty, flushed and moderately prostrated, complaining of excruciating headache. There were no findings of importance except that her temperature was 102.5 degrees Fahrenheit.

Laboratory Tests.—Blood: Hemoglobin, 90 per cent; red blood cells, 4,110,000; white blood cells, 7,800; 62 per cent polymorphonuclears; 26 per cent small lymphocytes. Urine: Specific gravity, 1.015; acid; trace of albumen; sugar, negative; Kahn, negative. Widal, negative. B. melitensis, negative agglutination.

Course of Disease.—The patient became afebrile on July 14, and was discharged August 1, 1935. At no

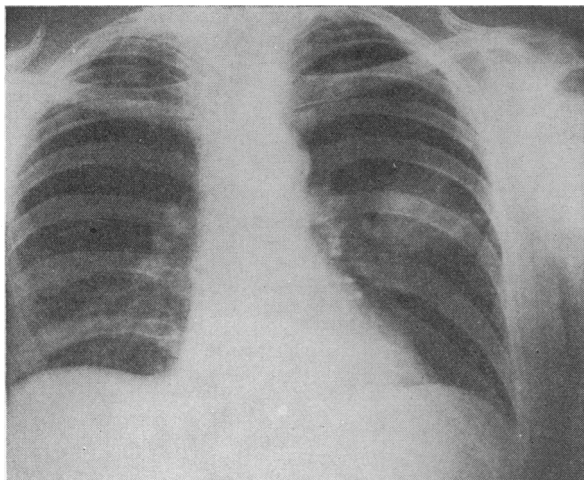


Fig. 1

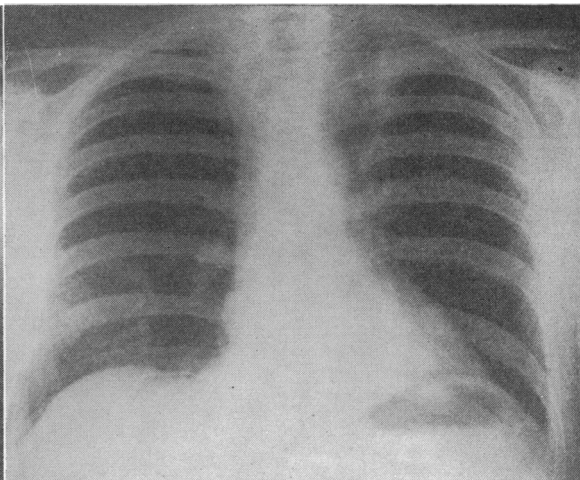


Fig. 2

Fig. 1.—Roentgenogram of lung fields showing annular area of pneumonic consolidation in left lung.

Fig. 2.—Roentgenogram of lung fields nine days later, showing absence of lesion in left lung and presence of similar lesion in lower right lung.

time was there any sputum obtained, although a slight hacking cough was present at times. Nasopharyngeal washings failed to infect mice.

EPIDEMIOLOGY

Epidemiology.—Many birds were present in the store when Mr. C. T. C. became ill June 14, 1935, including thrushes, mocking birds, redbirds, bulbuls, Petz conures, canaries, African love birds, and one Amazon parrot. Only one bird was ill, a Petz conure. This bird had been in the shopkeeper's possession for many months, having come from a dealer in Laredo, Texas. It was a source of pride to the owner that he was able to keep this sickly bird alive so long.

On June 22, Dr. K. F. Meyer of the Hooper Foundation, en route through Santa Barbara, took this bird

with him to San Francisco. After exhaustive bacteriologic investigation, the organs of this bird were pronounced free from psittacosis virus.

Following this result, scrutiny of the shop drew attention to the Amazon parrot (*Amazona barbadensis* Gmelin) and, while in fair condition, suspicion warranted sacrifice of the bird. Bacteriologically, the organs were sterile, but inoculations of mice with emulsions of the liver, spleen, and kidneys produced lesions of psittacosis in the rodents. The virus readily passaged.

This bird, contrary to earlier statements of the manner of purchase, had been obtained from an itinerant peddler within recent weeks. At the time of purchase the cage from which the bird came contained one dead and two live parrots.

COMMENT

Luckie has summarized the clinical manifestations and important factors in confirming the diagnosis from studies made in this country following the epidemic of 1929-1930. Roth,¹ in discussing Luckie's paper, has outlined the restrictions by the United States Public Health Service placed on imported birds and the quarantine regulations affecting the same. These have become more stringent with each successive outbreak since the Surgeon-General issued his first order on January 25, 1930, affecting parrots entering the United States from a foreign port. It is possible that more careful regulations affecting interstate and intrastate traffic in birds of the psittacine family are required. The repeated warnings of Meyer^{2,3} with respect to protective measures have borne fruit, as demonstrated in a recent survey of 164 aviaries by the Department of Public Health in which 55 per cent yielded, by repeated examinations, parakeets which were free anatomically and by mouse tests of latent psittacosis.

The roentgenologic diagnosis of pulmonary lesions offered the first definite evidence of the nature of the disease. The migrating pneumonic process mentioned by Meyer³ is beautifully demonstrated by the roentgenograms taken nine days apart. In Figure 2, the lesion in the left lung (Figure 1) had completely disappeared and now

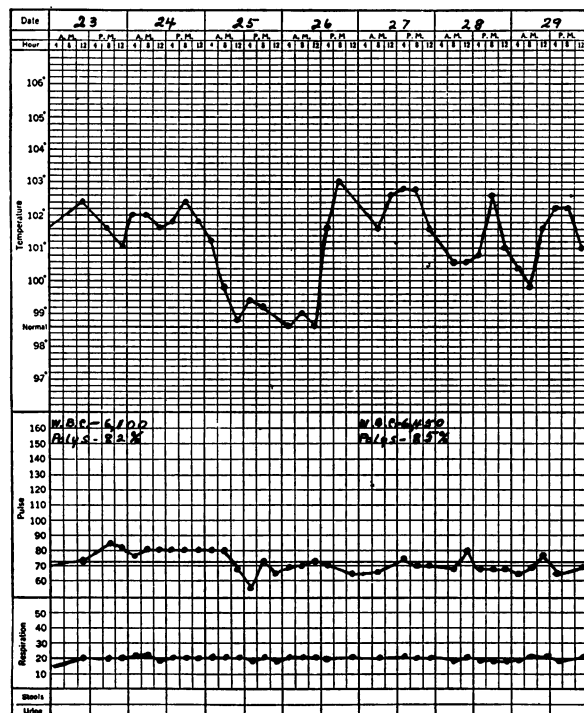


Fig. 3.—Showing fever curve with normal respiration rate and pulse rate curves; mild leukopenia present.

an annular shadow is present in the lower right lung field. The pathology of the pulmonary lesions, as well as other organs in man, has been thoroughly described by R. D. Lillie⁴ and by Poloyes and Lederer.⁵

The marked febrile state with normal respirations and pulse rate, with mild leukopenia, are shown in Figure 3. The clinical manifestations and the similarity to and the variation from the influenza symptomatology are commented on by M. A. Rabinowitz and S. H. Livingston.⁶

CONCLUSIONS

1. Two cases of psittacosis have been presented from one of which the virus has been demonstrated from the sputum.

2. The symptom complex has been typical of this type of infection, except for the occurrence of severe abdominal pain manifested in Case 1.

3. The epidemiology has been established and traced to an Amazon parrot in the store of the owner (Case 1).

4. The purchase of this bird from an itinerant vendor proved the break in the link of the control of the sale of psittacine birds by the Public Health authority.

5. That more stringent regulations with penalties for the sale of birds that have not been inspected by public health authorities is in order within the confines of the State of California, so that the desired-for goal—no psittacosis in the American bird industry—may be reached.

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DISCUSSION

KARL F. MEYER (Hooper Foundation, Medical Center, San Francisco).—A memorandum giving a summary of investigations concerning two cases of psittacosis in owners of a pet-shop due to contact with an Amazon parrot [*Amazona barbadensis* (Gmelin) *Sive Amazona ochroptera* (Reichenow)] would be as follows:

May 10, 1935: Mr. J. T. Cr., owner of a pet-shop at Santa Barbara, bought an Amazon parrot (*Amazona barbadensis*) from an itinerant peddler. At the time of the purchase the cage contained one dead and two live parrots. He transferred the Amazon to a backroom at his store, where he had various other birds (thrushes, mocking birds, redbirds, bulbuls, and Petz conures); after June, 1935, he added white canaries and African love birds. The Petz conure was ill and required special attention.

June 21, 1935: Mr. J. T. Cr., who had been ill for a week and felt below par for a month, was attended

by Doctor Steele. He complained of fever, chills, general aches, pain, nausea, and vomiting.

June 23, 1935: On account of high temperature (103 degrees, pulse 80) and persistent nausea and vomiting, he was hospitalized. Irrational at times; white blood cells, 4,600; considerable mucopurulent sputum; sharply defined central area of consolidation in left lung.

June 26, 1935: Mrs. J. T., developed similar symptoms and was hospitalized. Nasopharyngeal washings of Mrs. Cr. obtained on July 19, 1935, failed to infect mice with psittacosis (bacteriologic washings contaminated with *B. coli* and alpha streptococci).

June 27, 1935: Sputum collected from Mr. J. T. Cr. (approximately thirteenth day of disease) infects mice with typical lesions of psittacosis on the ninth day. Bacterial flora mostly staphylococci and alpha streptococci.

June 29, 1935: Sputum collected (fifteenth day of disease) infects mice with psittacosis lesions.

July 4, 1935: Sputum collected (approximately nineteenth to twentieth day of disease). One mouse of four infected; remainder non-infected.

July 12, 1935: Normal temperature.

July 20, 1935: Sputum fails to infect mice to date.

Comments.—Examination of Petz conure (*Eupsittula canicularis* Linnaeus), killed by the daughter of the owner on June 29, revealed a slight catarrhal inflammation of the intestines. The organs were bacteriologically sterile and were free from psittacosis virus.

The Amazon parrot was chloroformed at the store on July 19, 1935. The autopsy revealed the following:

Immature male, in fair condition; anus slightly soiled; nasal openings dry, but the conchae were injected and moist. The abdominal cavity moist; the spleen enlarged (15 by 13 by 10 millimeters) and soft; the liver enlarged with rounded edges and friable; the kidneys dark and firm; the lung crepitant throughout; the air sacs slightly thickened.

Bacteriologically the organs were sterile, but on inoculation on mice the following results are noted:

Spleen: Mice died on the seventh day (one mouse, one killed) with lesions of psittacosis.

Kidneys: Mice died with lesions of psittacosis; + seventh day (two mice).

Liver: Mice died with lesions of psittacosis; + ninth day (one mouse, one survived, weak virus).

Cloaca content: Non-infectious for mice.

Nasal mucosa: Non-infectious for mice.

Summary and Suggestions.—1. A 65-year-old pet-shop owner and his 62-year-old wife, who have doubtless had in the past contact with infected shell parakeets, contracted psittacosis through exposure to an infected Amazon parrot. The clinical course was typical and the virus was demonstrated in the sputum of one patient.

2. The parrot was recently imported. It is not unlikely that the virus was primarily discharged in the urine and thus mixed with droppings at irregular intervals. Bird, at the time of examination, in latent state of infection.

3. The importation of Central American Amazon parrots into California should be prohibited.

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The observations made by Dr. Arthur B. Steele deserve some comment.

1. Contemporaneous publications on psittacosis convey the impression that the apparent immunity of bird breeders and pet-shop owners to psittacosis is particularly evident in those who have had contact with tropical birds for many years. It has been reasoned that continuous exposure to the virus may induce subclinical infections and subsequently permanent immunity. Nobody will deny the reasonableness of these conclusions, since psittacosis in the natural and the experimental hosts (mice, guinea-pigs) has a pro-

nounced tendency to latent infections. Furthermore, epidemiologic observations indicate that, as a rule, the beginners in the parakeet raising trade contract the disease. In fact, in California, 38 per cent of the reported cases of human psittacosis developed in the owners of large or small parakeet aviaries or in their families. Why these two pet-shop owners, who had handled sick and diseased birds for many years, failed to acquire complete protection is indeed an intriguing question. By comparison, it is evident that the two patients possessed a certain degree of resistance, since they recovered, irrespective of their ages (sixty-five and sixty-two). Furthermore, it is not unlikely that the resistance against psittacosis is probably not absolute, but is conditioned by factors concerning which very little is known. In fact, it is probable that the striking resistance of many young and middle-aged individuals who may handle infected psittacine birds with impunity is nonspecific. It may be the expression of a hereditary conditioned nonsusceptibility which may gradually diminish with age. The complement fixation tests indicated that Mrs. C., who had a relatively mild infection, elaborated abundant antibodies in her blood serum, while the husband, tested during his convalescence, produced no immune substances. With the aid of these and similar tests, it is anticipated that some of the mooted questions relative to the immunity in psittacosis may be clarified.

2. Epidemiologically the Santa Barbara cases furnished ample evidence concerning the unreliability of testimony collected during the illness of the parties involved. Four independent investigators had been assured that the pet-shop owner, who had voluntarily discontinued the sale of parakeets in 1932, had not purchased any tropical birds in recent months. Since he had nursed for several months a supposedly sick Petz conure, suspicion was directed to this bird and in a spell of hysteria the daughter killed the bird without affording the investigators an opportunity to study the clinical symptoms. Since this conure was found to be free from psittacosis, both anatomically and by animal tests, the pet-shop owner, who by that time had recovered, was interviewed again. He then admitted the purchase of the Amazon parrot from an itinerant peddler on May 10, 1935. At the time of the purchase the cage of the peddler housed two live and one dead parrot. Notwithstanding this fact the purchase was made. On June 20 (five weeks later) the patient, who had been ill for a week, consulted his physician.

3. Investigations by no means completed failed to establish the origin of the Amazon parrot and the source of the infection. It is not unlikely that the bird entered the country illegally.

4. At the time the Amazon parrot was autopsied (July 19, 1935, eight weeks after the onset of the illness) the cloacal content and the nasal mucosa proved to be noninfectious. However, comparative tests indicated a striking concentration of the virus in the kid-

neys. This and similar observations made on other birds strongly support the belief that the virus leaves the bodies of the birds through contamination of the cloacal content with highly infectious urine.

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Report of Case (Reference to Illness of Doctor Hasseltine, United States Public Health Service).—In addition to the above discussion of Doctor Steele's report of cases, another case of infection of special interest to Californians is reprinted from the *Journal of the American Medical Association* (issue of August 31, 1935, page 727).

Owing to lack of funds, the State Board of Health found it necessary to close the psittacosis laboratory in Pasadena. The United States Public Health Service had detailed Doctor Hasseltine to cooperate in the State investigations. The item referring to his illness from psittacosis follows:

DOCTOR HASSELTINE ILL WITH PSITTACOSIS FOR SECOND TIME

Dr. Hermon E. Hasseltine, United States Public Health Service, is ill in San Francisco with psittacosis. This is the second time that Doctor Hasseltine has had the disease, the first attack having occurred in 1930 in Washington, where he was making laboratory studies of the epidemic that then prevailed. For three years Doctor Hasseltine was in charge of the psittacosis laboratory of the public health service at Pasadena, which was closed several months ago. He was then detailed to San Francisco to make a study of bubonic plague. While he has not been in contact with parrots recently, it is believed he acquired the infection in Pasadena, July 10, from instruments used in previous studies, which he handled while packing them for shipment. He became ill July 25 and on July 28 was admitted to the marine hospital. He is now much improved. So far as the public health service is informed, this is the only instance of psittacosis occurring a second time of which there is record. In March, 1930, Doctor Hasseltine suffered a moderately severe infection with psittacosis apparently acquired at the National Institute of Health, although definite history of his contact with infected birds could not be traced. His illness occurred at the time that ten other persons connected with the institution were infected. Two of this group were in direct contact with infected birds, but the means of transmission of the disease was not determined in the other cases. Doctor Hasseltine has been with the public health service for twenty-six years.

PSITTACOSIS IN AUSTRALIA

By K. F. MEYER
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QUITE recently Dr. F. M. Burnet kindly sent a summary of his detailed studies on psittacosis established in the Australian parrots. In a previous publication (*The Medical Journal of Australia*, December 8, 1934, p. 743) he conclusively demonstrated that the red-backed parrots (*Psephotus*

TABLE 1.—*Psittacosis in Australian Parrots*

Genus and Species	Proved Psittacosis	Enlarged Spleen without virus	Normal Spleen	Total
Lorikeets (<i>Trichoglossus</i>).....	7 (58%)	2 (17%)	3 (25%)	12
Cockatoos (<i>Kakatoe</i>).....	3 (6%)	10 (21%)	34 (72%)	47
Cockatiel (<i>Leptolophus</i>).....	6 (60%)	0	4 (40%)	10
Rosellas (<i>Platycercus</i>).....	5 (5%)	19 (18%)	84 (78%)	108
Grass Parakeet (<i>Psephotus</i>).....	28 (41%)	0	40 (59%)	68
Budgerigar (<i>Melopsittacus</i>).....	1 (5%)	1 (5%)	19 (90%)	21
Grand total.....	50 (18.2%)	32 (12%)	184 (70%)	266